Another concern is that patients in the SE group presented a significantly higher incidence of more than mild aortic regurgitation, a well-known predictor of mortality after TAVR (5). However, the CHOICE trial failed to show any association between aortic regurgitation and mortality, possibly due to the previously described imbalance in baseline characteristics between the 2 groups.

Finally, patients in the BE group showed a trend for an increased rate of stroke compared with SE patients. Of interest, the authors reported an unexpected finding of 4 cases (3.4% of BE-implanted patients) of early prosthetic valve dysfunction in the BE group, possibly attributed to valve thrombosis, suggesting a possible link with the higher stroke rate. However, patients enrolled in the BE group at baseline compared with SE group also presented a higher incidence of atrial fibrillation, despite no difference in antithrombotic therapy, that might play a role in the higher stroke rate observed in the BE group.

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REPLY: Outcomes After Transcatheter Aortic Valve Replacement With Balloon-Expandable Versus Self-Expandable Valves

CHOICE Trial Results

We appreciate the thoughtful comments of Dr. Montone and colleagues concerning the 1-year clinical outcome of the CHOICE trial, but do not share their concerns about a differential impact of baseline characteristics on the observed mortality rates in a randomized setting. Among all baseline clinical and echocardiographic characteristics of the CHOICE population, only sex was statistically significantly different between the balloon-expandable (BE) and self-expandable (SE) groups (age and baseline left ventricular function were not significantly different) (1,2). As mentioned in the paper, we performed a logistic regression analysis to adjust for sex, and the results are essentially unchanged if sex is taken into account (2) (unadjusted p value for all-cause mortality at 1-year using the Fisher exact test = 0.37, adjusted p value for all-cause mortality at 1-year using logistic regression = 0.33).

The assumption of Dr. Montone et al. that more than mild prosthetic valve regurgitation was not associated with higher mortality in the CHOICE population is not correct. As briefly mentioned in the discussion section of our paper (2), device success (which was mainly driven by the absence of more than mild paravalvular leaks) was independently associated with improved survival at 1 year (adjusted odds ratio calculated by logistic regression = 0.16, 95% confidence interval: 0.04 to 0.67, p = 0.01). As previously discussed, the lack of a mortality difference between both devices despite differences in device success could be partially explained by the moderate sample size of this study as well as the numerically higher rate of thromboembolic events in the BE group, although this remains speculative.

Finally, a potential association between the numerically higher incidence of baseline atrial fibrillation (AF) and the occurrence of stroke in the BE group cannot be entirely excluded. In fact, 6 of 11 stroke events in the BE group occurred in patients with AF, but the rate of new-onset AF was nearly identical in the BE group (9.8%) and SE group (9.4%, p = 1.0) (2).

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Management of Noncompaction Must Consider Nongenetic and Myopathic Implications

We read with interest the review about isolated left ventricular hypertrabeculation/noncompaction (LVHT) by Hussein et al. (1). However, the paper raises a number of concerns.

We disagree that LVHT exclusively results from incomplete intrauterine compaction of the developing myocardium. LVHT can be acquired in neuromuscular disorders (NMDs) (2), in pregnant women, and in athletes and may disappear in single cases, which are strong arguments against the noncompaction hypothesis.

We also disagree with the statement that LVHT was first described by Grant in 1926 (3). His case does not meet current diagnostic criteria because trabeculations were predominantly seen in the right portion of the single ventricle because the basal segments were predominantly affected and because intertrabecular spaces communicated with myocardial and epicardial vessels.

Earlier than Engberding in 1984, Feldt reported in 1969 a biventricular bizarre spongy myocardial pattern in an autopsy case and Westwood (4) presented a figure of a case (case 3) unambiguously showing biventricular hypertrabeculation.

We also disagree with classifying LVHT as a genetic disorder for the following reasons: LVHT has been associated with >30 different mutated genes and even more chromosomal defects, thus rendering it unlikely to be responsible for the same morphological abnormality; only a limited number of mutation carriers present with LVHT (LVHT often does not segregate with a mutation); a specific mutation associated with LVHT in a family member may be associated with variable cardiac abnormalities in another family member; LVHT can be acquired.

Application of the LVHT mass as a cardiac magnetic resonance imaging criterion is misleading because this parameter does not differentiate between mass of intertrabecular tissue (blood) and mass of trabeculations (myocardium). Because the relationship between these 2 figures varies considerably between patients, LVHT mass does not appear to be a reliable diagnostic marker.

When considering transplantation as a treatment of intractable heart failure, myotoxic immunosuppression should be avoided in patients with an NMD to prevent further neurological deterioration.

Overall, uncertainties about diagnosis, treatment, and prognosis of LVHT may remain unsolved as long as the pathogenetic background of LVHT is not fully elucidated.

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